evidence can be found to support other theories that have been advanced, such as that enlargement is the result of sexual excesses or sexual starvation, or that it is in the nature of a compensatory hypertrophy. The same is true of the observation that prostatic enlargement is a disease which has a definite geographical, or to be more exact, ethnographical distribution. If we could explain why it is so exceedingly rare amongst Mongolians we should be in possession of a valuable key to its etiology.

ACCIDENTS AFFECTING THE TESTICLE

Next to the prostate, the part of the genital tract that is most likely to suffer during the course of involution is the testicle. It is during this period of life that hydroceles are particularly liable to develop. Such hydroceles have up to the present been termed "idiopathic," since no satisfactory explanation of their etiology can be given. In my opinion a better term would be "degenerative," as they are almost always secondary to degeneration in the testicle itself. It has, of course, long been recognized that the testicle associated with a hydrocele in elderly men is often hard, and a microscopical examination of the gland invariably reveals fibrosis associated with degenerative changes in the tubules and ducts. These changes are patchy in distribution; that is to say, one finds areas that are comparatively normal and adjoining areas where the epithelial cells are degenerating or have even disappeared. When we consider the testicle rather than its coverings we find that the epididymis is more likely to suffer during this period of involution. Clinically, this is shown by the development of multiple retention cysts, due to stenosis of the epididymal canal and vasa efferentia from the contraction of surrounding fibrous tissue. Mr. Ogier Ward states that in a study of cases of multiple epididymal cysts he found the average age at which they occurred was 55, a fact which, in my opinion, points to their being degenerative phenomena. They may indeed be considered analogous to the cysts so commonly found in the ovary at the time of the menopause. As a rule, they cause only a trifling discomfort to the patient, but since they result in an increase in size of the testicle they are not infrequently the cause of unnecessary anxiety. The simplest method of treatment, if treatment be required, is tapping, but if this only produces temporary relief, excision may be carried out.

Although the seminal vesicles show marked signs of degeneration in cases of prostatic enlargement, any symptoms for which they may be responsible are so overshadowed by the symptoms resulting from prostatic obstruction that they are negligible. Sometimes, as a result of obstruction in the ejaculatory ducts, the vesicles became dilated or pouched, and subsequently these dilated vesicles are often the site of a chronic inflammation. Occasionally a stone is formed from the deposition of lime salts on a nucleus of epithelial cells and spermatozoa. These vesicular concretions may give rise to such symptoms as painful ejaculation, pain on defaecation, and haemospermia. Their chief importance, however, lies in the fact that they may be mistaken for malignant nodules and lead to an error in diagnosis.

TREATMENT OF DISTURBANCES AT THE CLIMACTERIC

The treatment of the male climacteric consists in dealing separately with the various disturbances to which it gives rise. In the case of a woman it is otherwise, for not only is her menopause definitely genital in character, but we are fortunate in possessing various extracts of a proven efficiency. It is doubtful whether, even if we possessed them, similar benefits would result from the use of active testicular extracts in man. I have observed no benefit after attempts to increase the supply of testicular hormone by tying the vasa; nor have such blind efforts in endocrine therapy as the giving of prostatic extract to cases of enlargement borne any fruits. In fact, nothing I have seen leads me to believe either that the prostate has any internal secretion of its own or that its enlargement is associated directly with changes in any of the endocrine hierarchy other than the testis. Until, therefore, we understand more fully the factors responsible for prostatic enlargement, and until we are able to postpone in man the arrival of his critical age, we shall be compelled to deal with the various accidents that accompany it by the crude, but nevertheless successful, methods of the surgeon.

BIBLIOGRAPHY

Metchnikoff: *Études sur la nature humaine.* Paris, 1905
Maranon, G.: *The Climacteric.* London, 1929.
Walker, Kenneth: Nature and Cause of Old-age Enlargement of the Prostate, *British Medical Journal,* February 25th, 1922.
Lowsley, O.: *Surg., Gynecol. and Obstet.*
Ciechanowsky: *Anatomical Research on Prostatic Hypertrophy,* (Translated by Green), 1903.

OTITIC HYDROCEPHALUS: A REPORT OF THREE CASES

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In each of the following cases the clinical evidence suggested, up to a point, the presence of a cerebral abscess secondary to otitis media. The question at issue, when the patients were first seen by the writer, was whether an exploratory operation should be undertaken. In all cases the application of certain criteria led to the diagnosis of otitic hydrocephalus; symptoms of increased intracranial pressure were relieved by lumbar puncture, and the patient made a complete recovery.

Of the pathological basis of otitic hydrocephalus we have no precise knowledge. It is a condition in which otitis media—with or without such complications as a preliminary meningitis, extradural abscess, or lateral sinus thrombosis—is associated with an excess of normal cerebrospinal fluid under excessive pressure. The clinical symptoms are those of increased intracranial pressure—headache, vomiting, and papilloedema—and in this respect are similar to those caused by a cerebral abscess. There are, however, certain points of clinical distinction between the symptoms of otitic hydrocephalus and cerebral abscess upon which the differential diagnosis may provisionally be made at the bedside. When this stage has been reached the diagnosis may be confirmed by appropriate investigation of the cerebro-spinal fluid. The main points of clinical importance are as follows:

1. The most striking feature of otitic hydrocephalus is swelling of the optic discs. This, with haemorrhage and exudate, frequently amounts to several dioptres. In cerebral or cerebellar abscess, on the other hand, the optic disks commonly show no more than a slight venous engorgement. Such a degree of swelling as is met with in otitic hydrocephalus is rarely seen in a case of abscess, and is then usually accompanied by extreme drowsiness and malaise.

2. In striking contrast to the appearance of the fundi in otitic hydrocephalus is the general condition of the patient. Between the attacks of headache and vomiting he is likely to be alert, lively, and of good appetite. The patient with an abscess, on the other hand, generally looks and feels ill, is dull, constipated, and without appetite. This point of distinction may be blurred or lost when some other condition, such as a lateral sinus thrombosis, is associated with otitic hydrocephalus.

3. The common situations for abscesses are temporal and cerebellar. By the time symptoms of increased intracranial pressure are evident, a cerebellar abscess will have given rise to definite "localizing signs," of which the most important are nystagmus towards the site of the abscess, and inco-ordination in the use of the upper limb on the same side. A left temporal abscess also (in the case of a right-handed person) will almost certainly have produced localizing signs in the form of "word forgetfulness." Weakness of the opposite face and a homonymous defect in the opposite visual field, are also signs of localizing value in temporal lobe abscess. A right temporal abscess, however, is relatively silent, and the differential diagnosis from otitic hydrocephalus is more difficult.

If on the basis of these clinical points of distinction the condition of hydrocephalus is suspected, lumbar puncture should be done, a manometer such as Greenfield's being employed. The characteristic features in hydrocephalus are a high pressure and a normal fluid—that is, *no* excess of cells or protein. Fluid from a case of cerebral abscess is also likely to show a high pressure, but this is not so high as in hydrocephalus. Taking the average normal pressure as 150 mm. H_2O, the pressure in abscess may be from normal up to 250, while in hydrocephalus it is more commonly in the neighbourhood of 300. Finally, so far as I am aware, the fluid in a case of cerebral abscess is never normal. Usually there is a small excess of cells (mainly lymphocytes with a few polymorphs); always there is an excess of protein.

The diagnosis being settled by these means, the condition of otitic hydrocephalus will clear up if treated by lumbar puncture. Enough fluid should be removed on each occasion to bring the pressure to normal, and the procedure should be repeated so long as evidence (symptomatic or manometric) of increased pressure persists. Any local condition such as mastoiditis or lateral sinus infection should be treated on its own merits, but the existence of hydrocephalus is not in itself an indication for operative intervention. The conclusions thus stated were reported more fully in a paper by the writer in *Brain*[1] of this year. The cases now to be related have been encountered since that time.

CASE I

Doris S., aged 5. Under the care of Mr. T. B. Layton, Guy's Hospital. Seen September 13th, 1931.

The patient was admitted with symptoms of right-sided mastoid infection three weeks ago. Mr. Layton operated next day, and found dense bone with a bead of pus in front of the lateral sinus. At this time some neck stiffness was noted, but disappeared later. The temperature settled down, but has gradually risen again. There has been a good deal of complaint of frontal headache. Yesterday the patient had a rigor.

Examination.—Child alert, fretful, but later co-operated excellently. Right-handed. No speech defect. Optic disks show papilloedema 4 dioptres on right, 1 dioptre on left. Visual fields—full to rough tests. Cranial nerves—no defect. No inco-ordination or weakness. Deep reflexes—present. Abdominals—present and equal. Plantar responses—flexor. No neck stiffness. Heart and chest—normal.

Provisional Diagnosis.—Lateral sinus thrombosis and otitic hydrocephalus.

Lumbar Puncture.—Clear fluid. Pressure between 250 and 300 mm. H_2O. Examination: No cells. Protein 0.02 per cent. Operation the same day showed lateral sinus thrombosis, which was dealt with.

September 16th, 1931.—Papilloedema subsiding. Child better.

September 23rd, 1931.—Condition excellent. Temperature subsiding. No headache. Papilloedema 1 dioptre right; 1/2 dioptre left. Subsequent recovery complete.

CASE II

William C., aged 26. Under the care of Mr. T. B. Layton, Guy's Hospital. Seen June 24th, 1931.

History.—April, 1931, acute otitis media followed by right-sided headache. May 14th, 1931: Admitted to hospital. May 21st, 1931: Mastoid operation by Mr. Layton. Extensive bony disease. Dura and lateral sinus normal. May 24th, 1931: Sudden rise of temperature to 104°. Further operation showed lateral sinus partially thrombosed. Jugular vein tied. Anti-streptococcal serum given. Subsequently temperature gradually subsided, and, after June 15th, 1931, remained normal. General condition good, but patient complained of severe bifrontal headache on waking in the morning, and of failing vision.

June 24th, 1931.—Mentally clear. Speech normal. Optic disks—4 dioptres of papilloedema right and left, with haemorrhages and exudate. Visual fields—full. Cranial nerves—slight weakness, of peripheral type, right face (ascribed to operation). No other defect. No weakness or inco-ordination. Deep reflexes—present and equal. Abdominals—present and equal. Plantar responses—flexor.

Provisional Diagnosis.—Otitic hydrocephalus.

Lumbar Puncture.—Clear fluid. Pressure 312 mm. H_2O. Examination: No cells. Protein 0.02 per cent. Further lumbar punctures were done at increasing intervals of time until the pressure became normal.

July 11th, 1931.—Papilloedema less. Occasional slight headache. Left hospital later recovered.

CASE III

Winifred B., aged 18. Under the care of Mr. Nicol Rankin, Central London Throat, Nose and Ear Hospital. Seen March 12th, 1931.

History.—Admitted February 24th, 1931, with a story of right otorrhoea for two months and complaint of headache for the past week, with vomiting after every meal. Tenderness over right mastoid antrum. February 25th, 1931: Operation by Mr. Rankin—right Schwarzte mastoid operation. March 10th, 1931: Rigor, followed by rise in temperature. Right-sided tinnitus and occasional twitching right side of face.

March 12th, 1931.—Mentally clear and co-operative. Speech normal. Right-handed. Optic disks—papilloedema 5 dioptres right and left, with haemorrhages and exudate. Visual fields—full to rough tests. Cranial nerves—nystagmus elicited on conjugate deviation to left. Slight weakness right orbicularis palpebrarum. Other cranial nerves normal. No weakness or inco-ordination. All deep reflexes present and equal. Abdominals—present and equal. Plantar responses—flexor. No neck stiffness. No signs elsewhere.

Provisional Diagnosis.—Otitic hydrocephalus associated with lateral sinus thrombosis and labyrinthitis.

Operation by Mr. Rankin the same day revealed lateral sinus thrombosis, which was dealt with.

Lumbar Puncture.—Clear fluid. Pressure not measured. Examination: No increase of cells or protein.

The patient made an uninterrupted recovery, and was reported by Mr. Rankin, on May 3rd, as perfectly well.

There are two points in the story of these cases which deserve further comment. One is the occurrence in all three of lateral sinus thrombosis. It has been suggested by Leidler,[2] arguing from the experience of a single case in which the same association was observed, that hydrocephalus may result from lateral sinus thrombosis on account of obstruction to the venous outflow from the cranium. Against this hypothesis it may be said that, if it were true, hydrocephalus should be a frequent instead of a rare accompaniment of venous thrombosis. Moreover, among the cases of otitic hydrocephalus that have so far been published there have been several in which no evidence of lateral sinus thrombosis has been present. The second point of interest is the age in the second case described above. It would appear from the cases so far recorded that otitic hydrocephalus is rarely met with in adults.

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REFERENCES

[1] Symonds, C. P.: *Brain*, 1931, liv, 55.
[2] Leidler, R.: *Journ. Laryng. and Otol.*, 1928, xliii, 672.